BINOCULAR HEMIANOPSIA AND OPTIC NERVE ATROPHY IN A CASE OF DIABETES MELLITUS.[1]

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On November 22, 1900, Dr. Cunningham, of Vineland, New Jersey, referred to the Eye Department of the Jefferson Hospital, Mrs. E. W., aged 55, on account of failing vision. He wrote that the patient's vision had been decreasing for the past 6 years and that he had been unable to find that the cause lay in an error of refraction. At the time of her visit vision equalled R. 20-200; L. 20-200, not improved by glasses. The ophthalmoscope showed clear media and advancing atrophy of the optic nerves. The retinal arteries and veins were diminished in size. There were no patches of exudation or of hemorrhage in the retina. The pupils were equal and responsive, sluggishly to light, actively to accommodation. Dr. Cunningham reported that the patient had been suffering from diabetes mellitus many years, but for some weeks the urine had been free from sugar. The quantity in 24 hours did not exceed the normal. The percentage of sugar at the height of the disease had varied from 6 to 8%. He had regulated her diet and administered codeine with the result of causing the disappearance of the sugar.

The most remarkable ocular symptom in this case was the peculiar limitation of the fields. They were limited in all directions and were irregularly hemianopic, with detached scotomata. The perimetric measurements were repeatedly made and with great care upon the hospital perimeter. Figs. I and II are the charts made with white and colored test objects 5 mm. square; the larger boundary indicating limits for white, the smaller for colors.

The atrophy of both nerves had advanced to about an equal degree, both fields were irregularly hemianopic and showed considerable difference in their forms; the boundary line between the seeing and the blind fields was sharply outlined by the one candle power, 1 cm. electric light.

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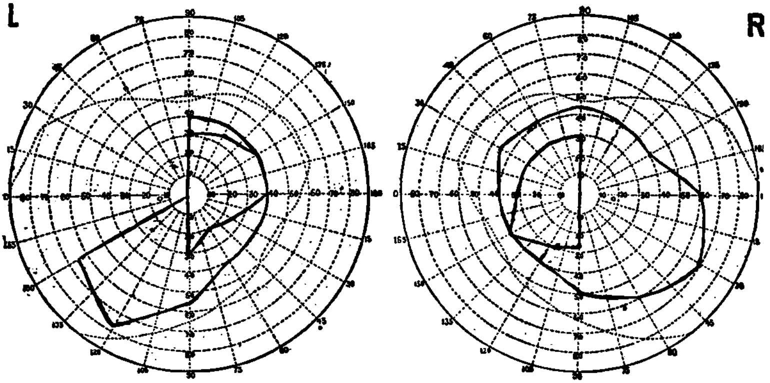
[1] Read before the Section of Ophthalmology, College of Physicians of Philadelphia, Feb. 19, 1901.

The patient was referred to Dr. Dercum, of the Neurological Department, who reported probable disease of the 4th ventricle, basing this diagnosis upon the diabetes and upon the ophthalmic report which was confirmed by repeated observations. The urine report made December 6: Reaction very acid, specific gravity 1034; urea 1¾%; sugar present by Fehling's, Boettger's and Trommer's test; quantitative estimation of glucose shows 3 gr. to the fluid ounce, or about ½%; no casts; no albumin.



Figs. I and II. Charts made with white and colored test objects, 5 mm. square; the larger boundary indicating the limits for white, the smaller for colors.

The ocular complications of diabetes are so varied and the literature so extensive that it is beyond the limits of a clinical paper to give more space than that required for simple enumeration, excepting the complications especially illustrated by the reported case. Lawford, in Norris and Oliver's System, describes them in the following order: Failure of accommodation; mydriasis; increase of refraction; paralysis of ocular muscles; keratitis, iritis and iridocyclitis; cataract; retinitis and disease of the optic nerve.

Optic nerve disease has been described by Leber[2] under three headings: (1) Amblyopia without ophthalmoscopic changes with or without limitation of the field; (2) Atrophy; (3) Hemianopsia. He believes in the few cases of hemianopsia reported the defective vision was not due to the glycosuria but to the localized intracranial disease. R. T. Williamson, in his work on "Diabetes Mellitus and Its Treatment," 1898, does not mention hemianopsia among the eye symptoms of diabetes. He describes at length the differential diagnosis between diabetic retinitis and albuminuric retinitis, and refers most of the causes of the defective vision to the retinitis, failure of accommodation and vitreous opacities. Galezowski,[3] reports the case of a man 58 years old with myopia of 5 D. who had suffered for two years with right homonymous hemianopsia that had suddenly appeared. He sought advice on account of diabetic keratitis. This patient also had anesthesia of the left cornea. The author questioned whether this complication was simply a symptom of paralysis of the 5th nerve or whether it also could be referred to an intracranial lesion. He was inclined to refer also the keratitis to a central disturbance. He collected 10 cases of diabetic keratitis of which this was the only one with hemianopsia.

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[2] Graefe Saemisch Handbuch, Vol. iv.

Atrophy of the optic nerve in diabetes is either primary or secondary to retinitis. In the former the loss of visual acuity is rapidly progressive until it is complete. The field shows contraction, sometimes concentric, sometimes irregular. The ophthalmoscopic appearances are those usual to simple optic nerve atrophy. Lawford[4] says the complication is extremely rare and the cases on record in which simple optic nerve atrophy was present are few in number and in several of them it is questionable if the optic nerve lesion should be considered the result of the glycosuria. "Of fifty cases with ocular symptoms, observed by Leber[5] there were fourteen, *i.e*, twenty-eight per cent., with optic nerve trouble." In two cases in which a post-mortem examination was made a tumor was found pressing on the optic chiasm in one, and in the other there was no cerebral lesion, but there was distinct degeneration of the kidneys. Williamson says it is stated that occasionally the retinal changes are associated with primary optic nerve atrophy and he gives an illustration of such a case. He also says that it is exceedingly rare that a central lesion has given rise to an optic neuritis and glycosuria in the same patient. In the examination of 140 diabetic patients, Schmidt-Rimpler[6] obtained evidence of retro-bulbar neuritis not traceable to the abuse of alcohol or tobacco in 34. In one case, distinct atrophy of the macular fibers of the optic nerve was found on microscopical examination. Wiesinger[7] describes in detail a patient 57 years old with plastic iritis who had amblyopia; the ophthalmoscopic appearances were those of simple optic nerve atrophy without other limitation of the field than color scotoma; and another case with recurring iritis, senile cataract, glaucoma, and finally optic nerve atrophy of the left eye, all of which symptoms he traced to diabetes.

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[3] Recueil de Ophtal., 1879.

[4] Norris and Oliver's System.

[5] Bericht der ophthal. Gesell., Heidelberg, 1896, S. 104.

Oscar Dodd[8] in an able article on diabetic retinitis has published a list of 47 cases with their references; among them I find some that bear on the subject of this paper, which I will briefly mention: No. 4, Haltenhoff,[9] duration of disease 5 years, grayish-white papilla not clearly marked; No. 10, Culbertson,[10] patient recovered but right eye terminated in atrophy of retina and optic nerve; No. 17, Legrange,[11] duration of disease 5 years, right eye edema of papilla with small hemorrhages around it, left eye papilla excavated and atrophied without increase of tension; No. 19, Legrange,[12] papilla swollen and irregular in outline; No. 25, Legrange,[13] optic nerve, left eye, atrophied, commencing atrophy of right optic nerve. Thus, among all the cases, he mentions only 4 that had distinct optic nerve inflammation or atrophy, and makes no comment whatever in the table upon the visual fields of these patients. He believes the optic atrophy to be due, in at least some of the cases, to embolus of the central vein.

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[6] Ber. d. ophthal, Gesell., Heidelberg, 1896.
[7] Arch. f. Oph., 1885, No. 2.
[8] Arch. of Oph., April, 1895.
[9] Zehender's klin. Monatsb. f. Augen, 1893.
[10] Detroit Lancet, 1882.
[11] Arch. d'Ophtal, 1887.
[12] Ibid.
[13] Ibid.

Nettleship[14] describes the pathology of ocular changes, in which but very few investigations have been made, as follows: Most of the changes were found in the blood vessels of the kidneys, spleen, liver and some of the smaller vessels of the brain. There was thickening of the retina in all its layers with numerous varicose swellings; the nerve fibers were slightly swollen and meshes formed by the separated fibers were partly empty and partly filled by swellings similar to those in the retina. In the other layers there was a chronic edema with hypertrophy of the connective tissue frame-work. The retinal arteries were greatly changed; thickenings were present between the epithelium and fenestrated membrane, contracting the lumen. The relative thickening was much greater in the very minute arteries than in those of larger size; the muscular coat was not generally affected; the veins were distended without alteration of their coats; the capillaries of all parts were distended with groups of aneurysms.

Mr. Lawford[15] made the microscopical examination of an eye in case No. 11, reported by Nettleship, in which the changes recorded were those mentioned above. Dodd says that edema and active changes of the retina and optic nerve are rarely present, and gives this as one of the chief distinguishing features between diabetic and albuminuric retinitis. Embolism of the retinal artery was present in two cases, thrombosis of the central vein in one case (de Wecker and Masselon). Juler[16] says "In the early stage papillitis is not a marked sign, but later it becomes very prominent."

It would seem from this imperfect review of the most recent writings, that optic nerve disease is a rare complication of diabetes, and that the inflammatory changes of recurring double iritis are the most common. Amblyopia without ophthalmoscopic changes, so often referred to by writers, is probably due in all cases to an intracranial lesion or is the earliest stage of the retro-bulbar neuritis which leads to atrophy. Hemianopsia, optic nerve atrophy and amblyopia, are probably but indications of the three stages of one affection, namely, retro-bulbar neuritis terminating in atrophy. The case above reported seems to prove the truth of this statement, because (1) the patient has rapidly failing vision; (2) an ill-defined but still unmistakable hemianopsia; and (3) double optic nerve atrophy. The opportunity of examining a patient in this late stage of diabetes without inflammatory changes or loss of transparency in any of the media, can rarely occur and the report of the case seems to me worthy to be placed on record.

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[14] Opthal. Hosp. Rep. Vol. ix., 1877.
[15] Trans. Oph. Soc. U. K., 1882.
[16] Handbook Oph. Science, 1893.